A Practical Approach to the Identification and Management of Sleep-Disordered Breathing in Heart Failure Patients

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KEYWORDS

• Sleep apnea • Obstructive • Central • Heart failure • Chronic • Systolic • Respiration
• Positive airway pressure

KEY POINTS

- Sleep-disordered breathing remains a prevalent comorbidity in patients with heart failure and is associated with poor outcome.
- Screening for sleep-disordered breathing should be performed routinely among heart failure patients because of high prevalence and etiologic association with many risk factors.
- Lifestyle modifications, restoring euvolemic state, and optimizing heart failure medical and device-based therapies are essential steps in the treatment of sleep-disordered breathing in heart failure patients.
- Treatment of obstructive sleep apnea in heart failure with continuous positive airway pressure was not associated with reduction in mortality or cardiovascular end points in a recent large randomized trial.
- Continuous positive airway pressure therapy should continue to be considered in heart failure patients with obstructive sleep apnea based on sufficient evidence for safety and effect on sleep quality and daytime function.
- There is no clinical evidence thus far to suggest any effective therapeutic option for central sleep apnea among heart failure patients. Although the roles of pressure support therapies for central sleep apnea in heart failure still require further investigation, an implantable phrenic nerve stimulator may offer a promising therapeutic option.

INTRODUCTION

Sleep-disordered breathing (SDB) is prevalent in patients with both systolic and diastolic heart failure (HF), representing the most common comorbidity and affecting between 50% and 80% of all patients.[1–3] SDB is classified further into obstructive sleep apnea (OSA), which is caused by intermittent obstruction in the upper airways, and central sleep apnea (CSA), which is defined as a periodic loss of central respiratory drive. Both OSA and CSA share a common pattern of

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repetitive hypoxemia and arousals resulting in sympathetic activation, oxidative stress, and sleep disruption. Therefore, both types of SDB have a negative impact on quality of life and cardiovascular function. It is well known that SDB treatment improves markers of cardiovascular disease and lessens their progression into HF. Furthermore, among HF patients, untreated SDB is independently linked to worse outcome. Although treatment of SDB is safe, readily available, and associated with improvement in cardiac function and quality of life of HF patients, data on improving outcomes are disappointing thus far. Despite the high prevalence of SDB in HF patients, the sleep and HF organizations have not provided treating physicians with practice guidelines to advise management of SDB in HF patients. This lack of guidelines has created large deviations in practice standards and explains why only a small fraction of HF patients receive adequate screening and treatment of SDB.[4]

This article provides a comprehensive overview of SDB in HF with focus on practical screening algorithms and simplified therapeutic approaches to help clinicians better recognize and treat SDB among their HF patients.

DEFINITION AND CLASSIFICATION OF SLEEP-DISORDERED BREATHING

SDB encompasses breathing irregularities that mainly take place during sleep. They are characterized by cyclic pauses of sleep with ensuing hypoxia followed by partial neurologic arousals. This frequent disruption in sleep architecture produces several deleterious neurohormonal and oxidative effects that extend beyond sleep hours to the waking time.

SDB events are classified into apneas and hypopneas. Apnea is a complete cessation of breathing and is further classified into obstructive or central based on the presence or absence of associated effort. Hypopneas are episodes of decreased air flow and are often difficult to further classify into obstructive or central. SDB is generally classified into OSA and CSA based on the dominant type of events. In other words, if more than half of the events were classified as obstructive, the SDB is classified as OSA. Similarly, CSA is diagnosed when more than half of the events are classified as central. In both OSA and CSA, the severity of SDB is judged by the Apnea Hypopnea Index (AHI), which reflects the number of apnea or hypopnea episodes per hour of sleep. Mild disease is defined as an AHI of 5 to 15 per hour, moderate as 15 to 30 per hour, and severe as $\geq$30 per hour. OSA is common in the general population and in HF compared with CSA, which is mainly found in advanced HF. Importantly, it is well established that patients with HF can shift between one type of SDB to another from night to night.[5] The coexistence of OSA and CSA in the same HF patient can also manifest in having high percentage of central events in a patient classified as having OSA. The prevalence of these mixed SDB cases among HF patients is not reported. In the HF population, it is not uncommon to see both types of SDB coexist in the same patient.

THE EPIDEMIOLOGY OF SLEEP-DISORDERED BREATHING IN HEART FAILURE

SDB is prevalent in the overall population and more so in patients with HF. In the general public, the incidence of SDB among middle-age adults is estimated to be 9% to 24%,[6] whereas patients with AHI of $\geq$5 accounted for more 24% among the elderly in one study.[7]

In patients with HF, the prevalence of SDB far exceeds that of the general population.[1,6,8] Earlier epidemiologic studies estimated a prevalence of 24% to 37% for OSA and 40% for CSA in patients with HF.[1,6,8] Other reports showed higher prevalence of OSA than CSA in patients with chronic HF.[9,10] Studies of hospitalized patients with acute decompensated heart failure confirmed a higher prevalence of OSA in the inpatient population.[11] This finding could be explained by the strong association of OSA and cardiovascular risk factors that manifest clinically during the decompensated state.[12–14] Additionally, the increasing incidence of obesity in the general population, and the relation between obesity, OSA, and cardiovascular disease, lends further explanation to the higher prevalence of OSA among HF patients. CSA, however, is considered mainly a disorder of advanced stages of systolic HF and its relationship to common cardiac risk factors is not as strong as that of OSA.[1,11]

The prevalence of OSA in diastolic HF is less studied than it is in systolic HF. However, SDB seem to affect both types of HF at a similar rate. In one report, AHI of 10 or more was present in 50% of patients with isolated diastolic HF.[15]

There is also significant sex variation in the occurrence of SDB with men being more affected than women. The exact reason for sex mismatch is still unclear but can be attributed to the android pattern of weight gain seen in men and also to the effects of testosterone on respiratory centers and upper airway musculature.[16]

Age is a major risk factor for OSA in the middle-age population. After the age of 60, sex and weight become less important predictors of OSA; this is